SPECIFICATION AMENDMENTS

After the title and before "FIELD OF THE INVENTION" on page 1, please add the following:

CROSS-REFERENCE TO RELATED PATENT APPLICATIONS

This application is the U.S. national phase of International Patent Application No. PCT/US03/06344, which was filed on February 27, 2003, and which claims the benefit of U.S. Provisional Patent Application No. 60/360,543, which was filed on February 27, 2002, and U.S. Provisional Patent Application No. 60/370,189, which was filed on April 5, 2002.

Please replace paragraph 0002 on page 1, with the following:

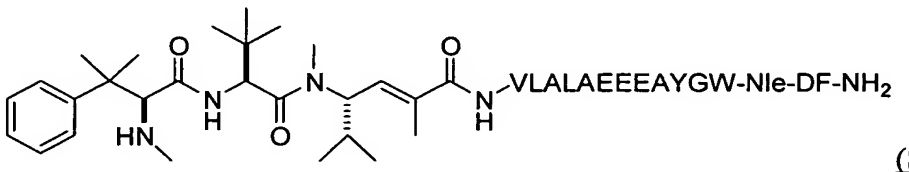
Systemic toxicity of drugs is one of the most serious problems of cancer chemotherapy and frequently is dose limiting. The appearance of the various classes of multiple drug resistance renders even good drugs ineffective by expelling them from tumor cells (Ling, *Cancer Chemother. Pharmacol.* 40: Suppl, S3-S8 (1997)). Various strategies have been used to get around one or both of these difficulties, but they still are among the most intractable problems of cancer therapy. Targeting of drugs specifically to tumor cells has been the goal of many studies. Various protein toxins conjugated to monoclonal antibodies directed to specific tumor antigens have shown some promise as drugs (Pastan, *Biochim. Biophys. Acta* 1333: C1-C6 (1997)), but severe problems, such as the development of neutralizing antibodies (Chen et al., *Gene Ther.* 2: 116-123 (1995)), have limited the effectiveness of the method. Another promising approach is to use cellular receptors for growth factors (Kihara et al., *Cancer Res.* 55: 71-77 (1985); Carpenter, *Curr. Opin. Cell Biol.* 5: 261-264 (1993); ~~Lemaristre~~ Lemaistre et al., *Breast Cancer Res. Treat.* 32: 97-103 (1994)), cytokines (Strom et al., *Annu. Rev. Med.* 44: 343-353 (1993); Waldmann et al., *Ann. Intern. Med.* 116: 148-160 (1992)), or hormones (Roth et al., *Anticancer Drug Des.* 10: 655-666 (1994); Rink et al., *Proc. Natl. Acad. Sci.* 93: 15063-15068 (1996)) as targets to deliver cytotoxic moieties to the receptor-bearing cells. In this approach, the receptor binds to a ligand that is conjugated to a toxic moiety, resulting in receptor-mediated endocytosis, wherein the ligand-toxic moiety conjugate is internalized, along with the receptor, by the targeted cell. Once inside the cell, the conjugate is susceptible to lysosomal proteases that cleave the linkage between the ligand and toxin, resulting in the release of the toxin from the conjugate. Through this approach, the delivery of a drug to specific cell populations can be achieved.

Please replace paragraph 0042 with the following:

Synthesis of cemadotin-peptide conjugates: Gastrin- linker merged sequences VLALAEEEAYGW(Nle)DF (SEQ ID NO: 25) and FLALAEEEAYGW(Nle)DF (SEQ ID NO: 28) were prepared by automated solid-phase peptide synthesis on ABI Rink amide resin (Applied Biosystems, Foster City, CA) utilizing ABI 433 peptide synthesizer (Applied Biosystems, Foster City, CA) equipped with conductivity monitoring system. Standard Fast Fmoc chemistry with HBTU/HOBt activation mixture was used (see Chan et al., *Fmoc Solid Phase Peptide Synthesis - A Practical Approach*, Oxford University, New York (2000)). Double-coupling was used for the last four N-terminal residues. The purity of the products was confirmed by analytical cleavage and LC/MS. Cemadotin with free carboxy terminus (27.6 mg, 0.05 mmol) in 0.5 ml NMP was activated by treatment with HOAt (3.2 mg, 0.025 mmol), CIP (12.4 mg, 0.045 mmol) and DIPEA (0.017 ml, 0.1 mmol). The mixture was added to 157 mg resin containing 0.274 mmol protected VLALAEEEAYGW(Nle)DF (SEQ ID NO: 25) per 1 g (0.045 mmol) in 0.5 ml NMP. The mixture was incubated overnight, washed with NMP, dichloromethane (DCM) and dried. Cleavage from the resin and precipitation with ether was performed as described above. The product was purified by HPLC on C3 reverse phase column (10 x 300 mm) in the gradient of 0.05% trifluoroacetic acid/water-acetonitrile. Calculated molecular mass: 2260.3. Found by LC/MS - 2260.2.

Please replace paragraph 0044 on page 14 with the following:

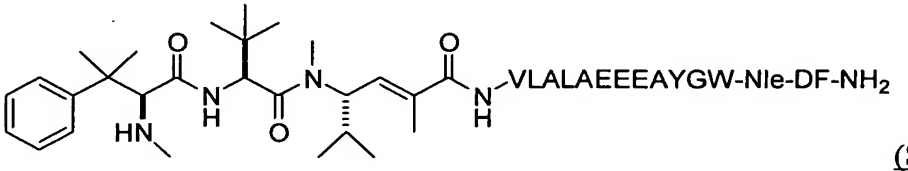
This example demonstrates the synthesis of conjugates comprising the hemiasterlin derivative, SPA110, and the gastrin decapeptide.



(SEQ ID NO: 26).

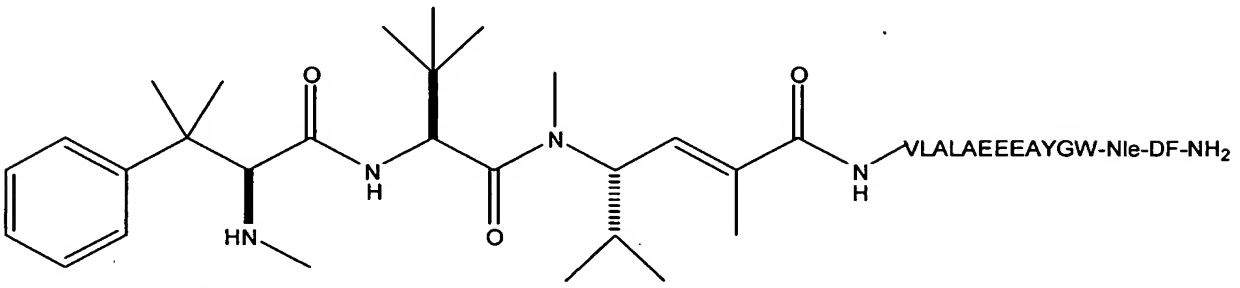
Please replace paragraph 0095 on page 24 with the following:

Preparation of MD025:



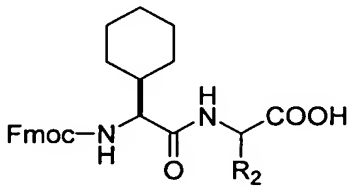
(SEQ ID NO: 26).

Please replace paragraph 0103 on page 25 with the following:
This example describes the activity of the conjugate comprising the hemiasterlin derivative, SPA110, the linker VLALA, and the gastrin decapeptide.



(SEQ ID NO: 26).

Please replace paragraph 0114 on page 27 with the following:
The dipeptide library of general structure



was prepared using preloaded Wang resins with suitable Fmoc-protected amino acid (0.1 mmol) on an ABI 433 peptide synthesizer (Applied Biosystems, ~~Froster~~ Foster City, CA). After Fmoc deprotection using 20% piperidine in NMP, 9-fluorenylmethoxycarbonyl-cyclohexylalanine was coupled using HBTU (2-(1H-benzotriazole-1-yl)-1,1,3,3-tetramethyluronium hexafluorophosphonate) and HOBt (N-hydroxybenzotriazole) as coupling reagents.

Please replace paragraph 0119 on page 28 with the following:
Upon testing on gastrin receptor-expressing 3T3 cells in accordance with the methods of Example 7, it was found that the most active compound of the series was the one in which R2 = Cyclohexyl (IC_{50} = 30 nM). The second most active compound of the series was the one in which R2 = Leu (IC_{50} = 120 nM). The gastrin conjugates potently and selectively inhibited the growth of gastrin receptor-expressing 3T3 cells. HTI-286-Cha-Leu-Ala-Leu-Ala-EEEAYGW-Nle-DF-NH_2 (SEQ ID NO: 27) had an IC_{50} = 10 nM (IC_{50} = 300 nM on nontransfected cells, which express a low-affinity gastrin receptor).